36. (New) A method of testing a compound for its ability to regulate transcription-activating effects of a peroxisome proliferator activated receptor-gamma (PPAR-γ), said method comprising assaying for changes in the level of reporter protein present as a result of contacting cells containing a GAL4 chimeric PPAR-γ receptor and a reporter vector with said compound;

wherein said GAL4 chimeric PPAR-γ receptor is introduced into said cells by a receptor expression vector comprising a DNA segment encoding at least the ligand binding domain of a PPAR-γ and a DNA segment encoding a GAL4 DNA binding domain, and

wherein said reporter vector comprises:

(a) a promoter that is operable in said cell,

(b) a GAL4 response element capable of being bound by said GAL4 DNA binding domain, and

(c) a DNA segment encoding a reporter protein,

wherein said reporter protein-encoding DNA segment is operatively linked to said promoter for transcription of said DNA segment, and

wherein said GAL4 response element is operatively linked to said promoter for activation thereof,

wherein an increase or decrease in the level of the reporter protein when said cells are contacted with said compound, relative to the level of the reporter protein when said cells are not contacted with said compound, is indicative of a compound that regulates the transcription-activating effects of said receptor.

37. (New) A method according to claim 36, wherein the DNA segment encoding said GAL4 DNA binding domain is introduced at the amino terminus of the DNA segment encoding said ligand binding domain of a PPAR-γ.

38. (New) A method according to claim 36, wherein the DNA segment encoding said GAL4 DNA binding domain is introduced at the carboxy terminus of the DNA segment encoding said ligand binding domain of a PPAR-γ.

39. (New) A method according to claim 36, wherein the DNA segment encoding the native DNA binding domain of PPAR-γ is substituted with the DNA segment encoding said GAL4 DNA binding domain.

40. (New) A method according to claim 36, wherein the DNA segment encoding said GAL4 DNA binding domain encodes amino acid residues 1-147 of the GAL4 protein.

41. (New) A method according to claim 36, wherein the DNA segment encoding said GAL4 DNA binding domain encodes amino acid residues 1-90 of the GAL4 protein.

42. (New) A method according to claim 36, wherein the DNA segment encoding said GAL4 DNA binding domain encodes amino acid residues 1-74 of the GAL4 protein.

43. (New) A method according to claim 36, wherein said compound is a putative antagonist for said PPAR-γ, and wherein said contacting is carried out in the presence of

increasing concentrations of said compound, and

a fixed concentration of at least one agonist for said PPAR-γ,

wherein a decrease in the level of the reporter protein when said cells are contacted with said compound and said agonist, relative to the level of the reporter protein when said cells are contacted with said agonist alone, is indicative of a compound that is an antagonist of said receptor.

44. (New) A method according to Claim 36, wherein said contacting is carried out in the further presence of at least one PPAR-γ agonist,

wherein an increase or decrease in the level of the reporter protein when cells are contacted with said compound and said agonist, relative to the level of the reporter protein when cells are contacted with said agonist alone, is indicative of a compound that regulates the transcription-activating effects of said receptor.

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45. (New) A method according to Claim 36, wherein said contacting is carried out in the further presence of at least one PPAR-γ antagonist,

wherein an increase or decrease in the level of the reporter protein when cells are contacted with said compound and said antagonist, relative to the level of the reporter protein when cells are contacted with said antagonist alone, is indicative of a compound that regulates the transcription-activating effects of said receptor.

H1

Please replace claims 16, 20, 27-29 and 33-35 with the following amended versions thereof:

16. (Thrice amended) A method of testing a compound for its ability to regulate transcription-activating effects of a peroxisome proliferator activated receptor-gamma (PPAR-γ), said method comprising assaying for changes in the level of reporter protein present as a result of contacting cells containing said receptor and reporter vector with said compound;

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wherein said receptor is introduced into said cells by a receptor expression vector comprising a DNA segment encoding PPAR-γ, and

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wherein said reporter vector comprises:

(a) a promoter that is operable in said cell,

(b) a hormone response element, and

(c) a DNA segment encoding a reporter protein,

wherein said reporter protein-encoding DNA segment is operatively linked to said promoter for transcription of said DNA segment, and

wherein said hormone response element is operatively linked to said promoter for activation thereof,

wherein an increase or decrease in the level of the reporter protein when said cells are contacted with said compound, relative to the level of the reporter protein when said cells are not contacted with said compound, is indicative of a compound that regulates the transcription-activating effects of said receptor.

20. (Twice amended) A method according to claim 16 wherein said compound is a putative antagonist for said PPAR-γ, and wherein said contacting is carried out in the presence of

increasing concentrations of said compound, and

a fixed concentration of at least one agonist for said PPAR-γ,

wherein a decrease in the level of the reporter protein when said cells are contacted with said compound and said agonist, relative to the level of the reporter protein when said cells are contacted with said agonist alone, is indicative of a compound that is an antagonist of said receptor.

27. (Twice amended) A method according to Claim 16 wherein said contacting is carried out in the further presence of at least one PPAR-γ agonist,

wherein an increase or decrease in the level of the reporter protein when cells are contacted with said compound and said agonist, relative to the level of the reporter protein when cells are contacted with said agonist alone, is indicative of a compound that regulates the transcription-activating effects of said receptor.

28. (Twice amended) A method according to Claim 16 wherein said contacting is carried out in the further presence of at least one PPAR-γ antagonist,

wherein an increase or decrease in the level of the reporter protein when cells are contacted with said compound and said antagonist, relative to the level of the reporter protein when cells are contacted with said antagonist alone, is indicative of a compound that regulates the transcription-activating effects of said receptor.

29. (Amended) A method of testing a compound for its ability to regulate transcription-activating effects of a peroxisome proliferator activated receptor-gamma (PPAR-γ), said method comprising assaying for changes in the level of reporter protein present as a result of contacting cells containing said receptor and reporter vector with said compound;

wherein said cells express native PPAR-γ, and

wherein said reporter vector comprises:

(a) a promoter that is operable in said cell,

(b) a hormone response element, and

(c) a DNA segment encoding a reporter protein,

wherein said reporter protein-encoding DNA segment is operatively linked to said promoter for transcription of said DNA segment, and

wherein said hormone response element is operatively linked to said promoter for activation thereof,

wherein an increase or decrease in the level of the reporter protein when said cells are contacted with said compound, relative to the level of the reporter protein when said cells are not contacted with said compound, is indicative of a compound that regulates the transcription-activating effects of said receptor.

H5 cont

33. (Amended) A method according to claim 29, wherein said compound is a putative antagonist for said PPAR-γ, and wherein said contacting is carried out in the presence of

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increasing concentrations of said compound, and

a fixed concentration of at least one agonist for said PPAR-γ,

wherein a decrease in the level of the reporter protein when said cells are contacted with said compound and said agonist, relative to the level of the reporter protein when said cells are contacted with said agonist alone, is indicative of a compound that is an antagonist of said receptor.

34. (Amended) A method according to claim 29, wherein said contacting is carried out in the further presence of at least one PPAR-γ agonist,

wherein an increase or decrease in the level of the reporter protein when cells are contacted with said compound and said agonist, relative to the level of the reporter protein when cells are contacted with said agonist alone, is indicative of a compound that regulates the transcription-activating effects of said receptor.

H6 cont.

35. (Amended) A method according to claim 29, wherein said contacting is carried out in the further presence of at least one PPAR-γ antagonist,

wherein an increase or decrease in the level of the reporter protein when cells are contacted with said compound and said antagonist, relative to the level of the reporter protein when cells are contacted with said antagonist alone, is indicative of a compound that regulates the transcription-activating effects of said receptor.